U. S. NAVAL SUBMARINE MEDICAL CENTER

Submarine Base, Groton, Conn.

REPORT NUMBER 585

DISCUSSION OF DISSEMINATED INTRAVASCULAR COAGULATION

IN DECOMPRESSION SICKNESS

by

LCDR John A. Holland, MC, USN

Approved and Released by:

J. E. Stark, CAPT MC USN
COMMANDING OFFICER
Naval Submarine Medical Center

20 June 1969

DISCUSSION OF DISSEMINATED INTRAVASCULAR COAGULATION
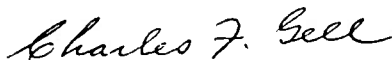# IN DECOMPRESSION SICKNESS

by

LCDR John A. Holland, MC, USN

SUBMARINE MEDICAL RESEARCH LABORATORY
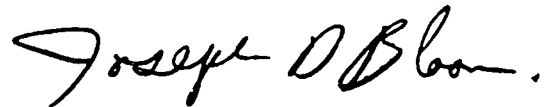NAVAL SUBMARINE MEDICAL CENTER REPORT NO. 585

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF099.01.01.03

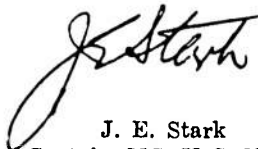Reviewed and Approved by:

Charles F. Gell

Charles F. Gell, M.D., D.Sc. (Med)
Scientific Director
SubMedResLab

Reviewed and Approved by:

Joseph D Bloom

J. D. Bloom, CDR, MC, USN
Director
SubMedResLab

Approved and Released by:

J. E. Stark

J. E. Stark
Captain, MC, U.S. Navy
Commanding Officer, NSMC

SUMMARY PAGE

THE PROBLEM

To examine the etiology of decompression sickness, and to reappraise the long-held bubble theory in the light of the concept of disseminated intravascular coagulation.

FINDINGS

An historical summary of the development of the bubble theory is given, as well as the points that have been debated and the reasons for the questions that have been raised. It appears questionable whether the bubble theory alone can explain all the observable phenomena. The concept of disseminated intravascular coagulation* is presented as a possible co-existing condition in at least some of the cases of decompression sickness. An extensive bibliography (124 items) is included.

APPLICATIONS

The information presented in this discussion is important for all medical officers and physicians who are or may be involved with the treatment of decompression sickness. It may lead to new forms of therapy.

---

*A transient coagulation occurring in the flowing blood throughout the vascular tree which may obstruct the microcirculation. It involves the transformation of fibrinogen to fibrin; it includes the agglutination of red blood cells, and sticking of platelets. It is often present in shock.

ABSTRACT

The etiology of decompression sickness is discussed from the point of view of its historical development from 1670 (Robert Boyle) to the present. A number of authors are cited who have called attention to circumstances unexplained by the bubble theory, and pointing to the existence of other mechanisms in the production of decompression sickness.

The concept of disseminated intravascular coagulation (DIC) is one of the mechanisms which has been receiving much attention as a possible co-existing condition in at least some of the cases of decompression sickness. DIC is a transient coagulation occurring in the flowing blood throughout the vascular tree which may obstruct the microcirculation. It involves the transformation of fibrinogen to fibrin; it includes the agglutination of red blood cells, and sticking of platelets. It is often seen in cases of shock.

Further development of these theories may lead to new forms of therapy for decompression sickness.

An extensive bibliography (124 items) is included.

DISCUSSION OF DISSEMINATED INTRAVASCULAR COAGULATION IN DECOMPRESSION SICKNESS

INTRODUCTION

The exposure of animals or man to decreased atmospheric pressures can provoke a variety of symptoms. In the extreme, decompression can lead to sudden and violent death. If properly controlled, however, decompression may be followed by a benign course with little or no observable pathology.

Historically, much has been written concerning variations in symptoms between caisson workers, divers, and aviators. It is the consensus, however, that any type of decompression can cause similar bodily derangements and that the number and severity of symptoms is generally proportional to the rate of decompression (assuming equivalent tissue saturations).

The bubble theory, by far, has had the widest support and has done much to elucidate the problems involved in decompression illness. It has certainly withstood the test of time. Nevertheless, the etiology of decompression sickness has been widely debated throughout history. Even today, it is still questionable whether the bubble theory alone can explain all the observable phenomena.

As an addition to the bubble theory, this paper will discuss the concept of disseminated intravascular coagulation (DIC) and its possible relevance to decompression sickness.

THE BUBBLE THEORY — THE HISTORICAL VIEW

As early as 1670, Robert Boyle[22] recognized the importance of bubble formation as a causative agent in the convulsive deaths of rapidly decompressed animals. Later investigators alternately supported or disputed the observations of bubbles in experimental decompression sickness. These inconsistencies seemed to be based on variations in one or more of the following:

1. Degree of tissue saturation
2. Rate of decompression
3. Size and type of experimental animals
4. Method and time of bubble observation—(autopsy, clinical or in vitro studies)

In decompression experiments carried out on rats, mice, cats, and rabbits, Hill and Greenwood (1907)[70, 71] found that small animals were relatively immune to decompression sickness.

In 1873, Bert[16, 17] observed that increased body fat seemed to lower the tolerance to decompression.

With respect to the above observations, it is known that smaller animals have a higher surface to mass ratio. Smaller animals also tend to have a higher metabolic rate and faster circulation times.[85] Teleologically, there is a direct correlation between the two on the basis of heat loss. It is also known that adipose tissue has a low metabolic rate, a relatively high solubility for inert gases, and a very meager blood supply.

If we may assume that the primary result of decompression is a consequential gaseous super-saturation of the blood and tissues then we can understand the vital role of circulation in attempting to relieve this abnormal situation. Thus, variations in bubble formation from one tissue to another and between one animal and another should be directly related to variations in blood flow.

Autopsy examinations have been notoriously inconsistent in their support of the bubble theory. Historically, this has been limited by incomplete exams and reports and also because post-mortem changes have obscured the picture to varying degrees depending on the time of autopsy.

In 1878, Heiberg[65] performed an autopsy within a few hours of death in a fulminating case of decompression sickness. According to the report, there were no signs of putrefaction and the skin of the chest, abdominal wall, and back were covered with multiple, livid, reddened patches. The skin was also emphysematous to the touch. On opening the

inferior vena cava, an extensive clot was extracted which was filled with trapped air bubbles of varying sizes. Air bubbles were also found within the intestines and pyramids of the kidneys contained air. No bubbles were seen in the brain or in the spinal cord on close examination, but a thrombus was found in the lower lumbar region.[74]

The above account is fairly typical of cases of violent decompression illness which are autopsied shortly after death. Schaffer,[108] however, has shown that the inconsistency of finding gaseous emboli can be explained on the basis that "increased activity of the heart and lungs may rapidly get rid of the gas bubbles in the blood so that few or none may be present at the time of death."[74] It is also possible in certain instances for some tissues to continue liberating gas into the circulation even after death. Again, there may be extreme variability depending on the tissue saturation and the circulatory status before, during, and after the actual moment of death.

In vivo methods of observing gas bubbles have partially resolved some of the controversy inherent in many of the post-mortem studies. As early as 1909, Hill[71,72] made direct observations of bubbles in the peripheral circulation of rapidly decompressed frogs and other animals. In 1945, Wagner[119] observed gas bubbles in the pial vessels of cats with the use of Forbes windows. Similar techniques as these have continued to variously reproduce intravascular bubbles in living specimens.[57,68,75]

Most of these studies indicate that decompression to one degree or another can and does liberate bubbles into the vasculature. According to Shilling,[112] however, the most important question is whether or not this liberation of gas is the etiological factor in decompression sickness.

CRITICISM OF THE BUBBLE THEORY

Accounts of intravascular bubble formation have dominated the literature of diving medicine for years. In most instances this has led to the "propter hoc, ergo hoc" assumption that bubbles are the etiologic agent in all cases of decompression illness. Indeed, there are hazards in this type of reasoning. Accounts have varied and there has often been insufficient evidence to explain the diverse symptomatology which typifies such cases. It remains that the most convincing argument in favor of the bubble theory seems to be the dramatic effect of recompression therapy.

There are a number of cases in the literature, however, in which patients have failed to show significant improvement after recompression treatment. Some cases involving decompression to altitude have actually shown an increase in symptoms. Rodbard[106] reviewed 14 such cases in 1946.

In an earlier paper,[85] the same author demonstrated that bends pain recurred at the same site upon decompression for periods up to four hours. These findings cannot be explained on the basis of the intravascular bubble theory. They do seem to support the possibility of extravascular bubbles. According to the literature, this is not a common finding. Heimbecker,[67] however, has recently demonstrated extravascular bubbles as an incidental finding in lethal decompression sickness.

One of the earliest criticisms of the bubble theory was that of Van Rensselaer in 1891[119] when he calculated that the quantity of gas liberated as a result of decompression was much less than the theoretical amount.

Swindle, in 1943,[115] enumerated a number of criticisms which have continued to be controversial. They are as follows:

1. It is difficult to explain the lag period (e.g., the period between minimum decompression and the appearance of symptoms). Sometimes there is a delay of several hours before symptoms appear.

2. Differences in susceptibility to decompression sickness are often marked.

3. Experiments in which large amounts of air (2,000 cc) have been rapidly injected intravascularly into animals without harm.

4. It is seldom possible to demonstrate bubbles in humans suffering from decompression sickness. When bubbles are demonstrated post-mortem, it has almost invariably been a case of explosive decompression and rapid death. It is warned that these bubbles may

not have been present before death.

Shilling[112] in 1941, also stated that there are a number of facts difficult to reconcile with the gas bubble theory. As did Swindle, he also criticized the often very long interval between decompression and manifestations of symptoms. Another discrepancy not covered by the bubble theory were cases which, after prolonged exposure to high pressures beyond the maximum calculated saturation time, it was impossible to desaturate safely according to the standard tables. In this regard, we are still having problems with decompression schedules in saturation dives to great depths. The problem of the "ideal" decompression model is yet to be solved.

In addition, Shilling considered variation in susceptibility in the same individual under identical working conditions, a difficult fact to accept. Recently, Coburn[31] has indicated a similar case of wide variation of symptoms in an individual subjected to identical exposures. He related that "Surely, in the same individual, almost identical amounts of nitrogen would be freed from the body tissues in each event."

Whitehorn, Lein, and Hitchcock[124] in 1947 subjected animals to explosive decompression. In 13 guinea pigs dying during such exposures, only 7 showed intravascular gas bubbles after recompression. These workers considered that intravascular gas bubble formation was a negligible hazard in explosive decompression. It seemed likely to them that anoxia was the prime cause of the fatalities in their experiments.

Heimbecker[67] has recently shown a surprising absence of gas bubbles in severe, non-lethal decompression sickness. He was able to observe the micro-circulation by in vivo technique in hamsters and dogs. From his experiments, he maintains that in severe lethal decompression sickness, gas bubbles are formed both intravascularly and extra-vascularly. Furthermore, only in those animals dying of decompression sickness could he ascribe the physiological derangements to gaseous emboli.
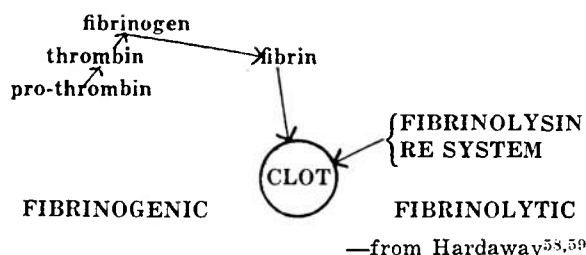
As evidenced by the success of recompression therapy, it does seem likely that bubble formation plays a significant role in the pathophysiology of decompression sickness. There probably are, however, other important mechanisms. New approaches in this regard are exemplified by Cockett.[32,37] He has recently shown that hemoconcentration is consistently observed in decompression sickness. Others such as Heimbecker, Chryssanthou, and Coburn have investigated forms of "decompression collapse" and have elucidated a number of possible mechanisms which may play a vital role. This emphasizes the complexity of the problem and the importance of new approaches in therapy. Thus, we cannot afford to accept the bubble theory too simply as an explanation of decompression sickness and place all our reliance on a single form of therapy, that of recompression.

WHAT IS DISSEMINATED INTRAVASCULAR COAGULATION?

According to Hardaway,[58,59] disseminated intravascular coagulation (DIC) is an acute, transient coagulation occurring in the flowing blood throughout the vascular tree which may obstruct the microcirculation. It may or may not result in an accumulation of fibrin, but it does involve the transformation of fibrinogen to fibrin. It includes the agglutination of red blood cells, and sticking of platelets. To understand these concepts, we will briefly review the theory of blood coagulation.

Current opinion seems to support the contention that coagulation and clot lysis is a continuum. It is assumed that fibrinogen (which has a half-life of approximately six days) disappears because of its conversion to fibrin. It is then removed by the fibrinolytic enzymes (plasmin or fibrinolysin) or by the reticulo-endothelial system. The speed of fibrin accumulation will depend on the concentration of thrombin, and the speed of fibrin dissolution on the concentration of fibrinolysin and the integrity of the reticulo-endothelial system. If fibrin is not removed with the same speed as it is formed, then an accumulation will occur. This equilibrium can be diagrammed as follows:

fibrinogen
thrombin → fibrin
pro-thrombin

{FIBRINOLYSIN
{RE SYSTEM

CLOT

FIBRINOGENIC   FIBRINOLYTIC

—from Hardaway[58,59]

According to Hardaway, changes in this equilibrium will result in DIC.

In his experimental model, Hardaway used intra-venous injections of thrombin, incompatible blood, and amniotic fluid in a number of dogs to provide similar results and thereby induced episodes of DIC. Characteristic, was the fact that these episodes could be prevented by pre-heparinization. The general features of experimental DIC are as follows: After injection there is an immediate fall in blood pressure (with occasional immediate death), but usually the blood pressure returns toward normal within 30 to 60 minutes. Occasionally there is a secondary fall in blood pressure and death within 24 hours. During the initial systemic hypotension there is alternate hypotension of the right atrium, vena cava, and portal systems. This is primarily due to the obstruction of the micro-circulation of the lungs, liver, and other tissues (consisting of thrombi formation in capillaries and secondary venospasm). The drop in the left atrial pressure then causes a decrease in cardiac output and decreased arterial blood pressure in spite of increased peripheral resistance due to thrombosis and venospasm. A bleeding tendency usually develops with capillary oozing, hyperpnea, increased peristalsis and frequently, urination and defecation with bloody diarrhea.

Hematologic changes consist of a fall in blood clotting elements including fibrinogen, prothrombin and platelets; the appearance of endogenous fibrinolysin and heparin-like substance; and a prolonged clotting time secondary to any or all of the above.

Typical pathologic changes consist of hemorrhagic necrosis of the G. I. mucosa; severe congestion of the abdominal viscera (liver, kidneys, etc.); and microscopic occlusion of capillaries and other small vessels by thrombi with secondary focal necrosis around the thrombi and hemorrhage. The thrombi consist of platelets and fibrin, but are predominantly congealed, packed and lysed red blood cells or white blood cells. The characteristic lung changes are not necrosis but effusion of blood and fluid into the alveoli.

It must be realized that the above is the purest example of DIC as reproduced under ideal laboratory conditions, Mechanisms of DIC are quite variable and have differing clinical and pathological features. Basic to all, however, is the characteristic widespread thrombosis (sometimes only evanescent) in small vessels and capillaries with secondary depletion of the clotting elements.

Hardaway describes numerous clinical states in which there is good evidence for DIC. These include shock, acute renal failure, hemolytic syndromes, syndromes of late pregnancy (e.g., eclampsia, defibrination syndrome), fat embolism, Shwartzman reaction, and extra-corporeal circulation.

In traumatic shock, DIC may occur by several different mechanisms which may operate simultaneously. These will be described below:

(1) With hemorrhage and trauma there is an increase in catacholamines and resultant arteriolar vasoconstriction. Accompanying this is a simultaneous opening of A-V shunts to the capillaries (80% of which are normally closed). The capillaries probably open by local histamine produced from mast cells secondary to anoxia. The volume of the vascular tree (but not the blood) may then double with resulting widespread capillary pooling. This markedly decreases capillary flow and leads to capillary and venous acidosis secondary to lactic acid accumulation. Slow-flowing, acid blood is hypercoagulable and together with thromboplastin (from hemolyzed red blood cells) there results widespread intracapillary coagulation. This consumes the clotting factors, halts perfusion, and may lead to cellular death and micro-infarction. Usually, the capillary clots are dissolved by endogenous fibrinolysin. If they remain long enough, however, focal tissue necrosis will occur. Often capillary flow is restored too late (to be effective) and cellular death superimposes in the area supplied.

Pathologic tissue examination of severe human shock cases have shown residual unlysed clots in capillaries of the lungs, kidneys, spleen, and intestines. In many cases routine microscopic tissue pathology will fail to find capillary thrombi in significant numbers. The main reason for this is the temporary nature of the thrombi.

In dogs given intravenous incompatible blood, thrombi were quite evident in renal capillaries 15 minutes later, but they rapidly decreased in number thereafter. The disappearance of the thrombi correlated with a simultaneous increase in endogenous fibrinolysin from 20 to 60 minutes.

(2) Although the capillaries and the microcirculation are the primary sites of vascular occlusion, DIC may occur in other areas throughout the vascular tree. Fibrin may deposit on any available surface, the largest one being the surface of red blood cells. This has been well documented in 32 cases of humans dying of shock. Some interesting observations in these cases were the uniform presence of rounded or ovoid globules. Some showed centrally located bubble-like areas. Smaller globules were shown by histochemical stains to be occupied by red blood cells in the center with outer fibrin coats. Larger globules with bubble-like centers seemed to be totally composed of fibrin with trapped or destroyed red blood cells. The mechanism of formation seems to be that under certain conditions red blood cells begin to accumulate fibrin. They then degenerate to form large globules or continue to circulate and add fibrin until they are large enough to cause small vessel occlusion. Coagulation then occurs behind and in front of the globule. The red blood cells disintegrate and release hemoglobin and thromboplastin to the periphery, thus perpetuating the event.

In dogs subjected to irreversible hemorrhagic shock, rouleau formation was observed. After 2 and ½ hours, agglutination of red blood cells occurred and many of the red blood cells took a fibrin stain. Blood studies showed a decrease in fibrinogen and platelets and prolonged silicone clotting times.

(3) Another mechanism is platelet agglutination. Injection of endotoxin or incompatible blood will cause widespread platelet agglutination. These gradually fuse together, obstruct small vessels and liberate thromboplastin. This causes fibrin accumulation at the site of the platelet clump or circulates and causes fibrin deposits on other surfaces (notably red blood cells as above). In addition, the thrombin is capable of causing further agglutination and destruction of platelets (this has been shown in vivo as well as in vitro).

Any or all of the above mechanisms may result in temporary or permanent occlusion of the micro-circulation. The process may start in the capillaries and extend directly; or platelets and red blood cells may accumulate, collect fibrin, and enlarge enough to occlude other areas of the micro-circulation.

The onset of DIC is heralded by the production of a clotting defect. This may come on quickly and within a few minutes. Because of widespread DIC, vast quantities of clotting factors are used up and the remaining blood becomes deficient. The coagulation defect may be evidenced by nothing more than a prolonged silicone clotting time. Of course this is most often missed because it is not usually checked unless there is clinical evidence of bleeding. In the extreme, the bleeding may be so great as to cause intractable fatal hemorrhage. The bleeding tendency is due to a decrease in any or all eleven of the clotting factors and platelets. The endogenous activation of heparin and/or the fibrinolysin system is the body's attempt to protect itself against this widespread clotting process.

Shock is a cardinal manifestation of DIC and occurs to a greater or lesser extent in all sysdromes of DIC. Shock has been defined as characterized by low arterial blood pressure or a disparity in volume between the blood and the vascular bed.

According to Hardaway it is possible (and in fact common) to have "shock" with normal or increased blood pressure. It is also possible to have "shock" with either cold, moist, pale skin or warm, pink, dry skin (e.g., arsenic poisoning or shock secondary to spinal anesthesia or vasodilators). It is said that shock is always reversible and that recovery takes place if transfusion is adequate. This

5

is usually true, however, patients die of shock even after apparently adequate blood replacement. There is general agreement that early shock is either due to decreased blood volume or increased vascular capacity or both. But, the big controversy is over so-called "irreversible shock," when there is seemingly no response to adequate blood replacement. In these cases the well-being of each individual cell depends on an adequate environment to furnish nutrition and carry away metabolites. This is determined by the composition of the blood and the adequate blood flow through the capillary serving the cell. In shock, this blood flow is disturbed. Shock can thus be defined as "inadequate capillary perfusion," and if this condition persists for any length of time it certainly may become irreversible.

DIC AND DECOMPRESSION SICKNESS

There are numerous cases in the literature in which shock is a significant feature of decompression sickness. According to Lamphier,[89] signs of shock have been noted in nearly 50% of tunnel workers with other symptoms besides pain. Most cases have occurred secondary to rapid decompression and many of them have demonstrated similar findings. Coburn[31] describes such cases as "Decompression Collapse Syndrome."

According to Hardaway,[58] in almost every type of shock (cardiogenic shock being a rare exception) DIC probably occurs to some degree. Very often it plays a significant role in the pathophysiological derangement.

As described in the previous section, intravascular agglutination is one of the mechanisms whereby an episode of DIC may be precipitated. Swindle[115] (1937) and End[44] (1938) maintain that agglutinated red blood cells may be the primary disturbance in compressed air illness, and that bubble formation may be looked upon as essentially a serious complicating factor. Their experiments showed that rapid decompression in animals caused exaggerated agglutination of cells within the blood vessels and even when it was impossible to demonstrate any gas bubbles, some of the animals showed vascular infarcts. Some of their experiments utilized in vivo techniques of observing the micro-circu-

lation. Furthermore, Swindle[115] demonstrated that CO_2 will cause an increase in the extent and duration of intravascular agglutination, whereas alkalization and O_2 administration tend to diminish or prevent this phenomenon just as they tend to diminish or prevent caisson disease. This correlates well with the increased incidence of bends seen with exercise.

Unpublished observations of End and Van Hecke[74] indicate that there is a fall in the CO_2 combining power of the blood during prolonged compression. This, according to these authors, may explain why the difficulty on decompression increases even after complete saturation with nitrogen (because of the increased tendency toward acidity of the blood which favors agglutination). End does not, however, believe that the importance of bubble formation can be denied and states that if severe, it may dominate the clinical and postmortem picture.

It may be that bubble formation is more intimately involved in this process than we realize. Any slowing of the circulation will tend to increase the likelihood of bubble formation as maximum circulation seems to be necessary in order to adequately remove gases from the supersaturated tissues. Thus, the delay (or lag period) often seen in bubble formation may be the period during which circulation is slowed because of intravascular agglutination.

Ecchymotic skin lesions and small vascular infarcts are a common finding in may cases of decompression sickness.[74] Shaffer[74, 108] describes these as "capillary echymosis." These lesions are often assumed to be secondary to direct obstruction of the vasculature by small bubbles. Often, however, they are probably due to non-gaseous emboli. These minute intravascular clots may be difficult to demonstrate because of the very active fibrinolytic system (see section IV). Swindle[115] has demonstrated non-gaseous emboli as a very common finding in animals subjected to decompression. In many of the same animals, gas bubbles were strikingly absent. He states that "fragile, non-gaseous emboli, apparently formed by flocculation of plasma, can be demonstrated by injecting the vessels with india

ink and dissecting out the emboli. The emboli are very fragile. Their location can be foretold by the behavior of the animal when it bites at or indicates discomfort in a certain part of the body. Injected ink flows only as far as the embolus and the pale area not filled with ink is an area of infarction. We have uncovered no new evidence which causes us to doubt that a non-gaseous embolus element is a profound factor in causing decompression sickness."

If DIC does play a significant role in decompression sickness it seems probable that coagulation studies should show some observable changes. Although bleeding tendencies have been clinically recognized and are not uncommon, very few coagulation studies have been done in conjunction with decompression sickness.[74, 97, 117]

In 1933, Aggazzotti,[2] at the University of Modena, Italy studied coagulation times in animals (dogs and rabbits) before and during decompression after exposures to pressures varying from 6 to 11 atmospheres. He found a shortening in coagulation time in most of the cases (12 out of 17) and an increase in coagulation time in 4 out of 17 animals. The increase in coagulation time seemed to occur in sick animals or those showing symptoms of decompression sickness. At first, these results seem difficult to explain. Nevertheless, Hardaway[58] finds that hypercoagulation often precedes an episode of DIC. These events may not always culminate in DIC, but they do predispose or "set the stage" for such an event.

In 1958, the French investigators Jullien, Leandri and Crozat[81] experimented on animals and man using the heparin test on animals and thromboelastography on man. They found blood disturbances, the most important being a tendency toward increased coagulation shown by the heparin test and the thromboelastogram which appears in both animals and man when decompression is too rapid. These disturbances occur very early and are seen as atmospheric pressure is reached. Because they occur even without perceptible symptoms they can be considered a pathological sign because, when rules of ascent are respected, no such phenomena are

seen. When there was an increase in CO_2 in the breathing mixture, the blood disturbances observed were more prominent.

Barthelemy[10] in 1963, found that coagulation was altered during rapid decompression in mice, rats, and rabbits. There was hypercoagulability in simulated dives to 30 meters and hypocoagulability at 60 meters. Because of earlier favorable results in using heparin as an adjuvant agent in recompression treatment in rabbits, it was also tried in five human cases. It was administered in doses of 50-100 mgms. twice a day. According to the authors, "spectacular improvements" were noted and in no instance was there any aggrevation of symptoms.

These same experimenters did observe bubbles in some of their animal studies. They maintained that "the bubbles increase blood platelet clumping, aggluination of erythrocytes and formation of plasma flocculates which provoke thrombosis.[10] Although they maintain that there is little risk of hemorrhage with the doses of heparin they employed, they recommend its use only for severe cases of decompression sickness in which the prognosis is bad and in which there is no improvement in spite of classic treatment.

Hardaway[58] states that exogenous administration of anticoagulants in cases of DIC have been disappointing. He gives the following reasons for this:

1. Heparin is effective only in preventing thrombosis, not in correcting it.
2. Heparin is inactivated in the presence of acidosis, a state which always accompanies DIC.
3. Heparin has no effect on platelet agglutination (sometimes an important initiator of DIC).

If given in time, it seems that heparin might be effective in some cases, in accordance with Barthelemy's results. Most often, however, by the time DIC is diagnosed, it is given too late to be of any value.

Many other adjuvant forms of therapy have been tried in decompression sickness.[28, 33, 91] Of these, Dextran seems to show the most promise. Recently, Cockett has shown

that plasma volume expanders have brought about dramatic recovery in cases of rapid decompression.[34, 36] He maintains that early institution of appropriate corrective therapy will lower the present mortality rate of 35% from shock which may accompany moderate to rapid decompression. According to him, this mortality rate has remained constant during the past 15 years.[36]

Cockett has consistently observed significant hemoconcentration with as much as 30 to 40% plasma deficits in both experimental and human cases of decompression sickness. He believes that plasma expanders act primarily by correcting this deficit. The exact mechanism which causes this plasma deficit has yet to be elucidated. It is suggested that inadequate microcirculation may be important. Heimbecker[66] maintains that vascular stasis plays a critical role in the mechanism of traumatic shock and it seems to offer an explanation for local and generalized fluid loss. The development of tissue edema in association with increasing red cell agglutination and stasis has been observed in vivo.[18] Knisely[86] has suggested that this is probably due to embolic blockage of small vessels by circulation clumps of erythrocytes producing capillary hypoxia and increased permeability.

According to Evarts[48] "low molecular weight Dextran exerts its greatest effect upon the peripheral circulation when there is an impairment by erythrocyte aggregation or by diminished perfusion pressures." Furthermore, he states; "The mechanisms by which low molecular weights Dextran exerts its influence upon the microcirculation are intimately associated with the pathophysiology of erythrocyte aggregation." He gives four basic mechanisms whereby low molecular weight Dextran appears to alter erythrocyte sludging and reduce blood viscosity:

1. It creates rapid plasma volume expansion which results in fluid dilution and a reduction in hematocrit value.
2. It may form a complex with plasma fibrinogen, and hence temporarily abate the known effect on viscosity by fibrinogen.
3. It has an affinity for the erythrocyte membrane and subsequently increase erythrocyte negativity and repulsion between erythrocytes.
4. It exerts a siliconizing effect upon injured walls of blood vessels, resulting in peripheral flow improvement.

With respect to the last point, it is interesting that Heimbecker emphasizes the use of siliconized clotting times in detecting episodes of DIC. Siliconized clotting times are more sensitive in view of the fact that the surface activating factors (factors XI and XII) are not "short circuited." These factors seem to have a significant influence on coagulation, as siliconized clotting times are twice to three times that of the standard Lee and White glass clotting times. It's possible that the surface tension in small intravascular bubbles found in decompression sickness may help activate factors XI and XII and thereby precipitate episodes of DIC.

Hardaway points out that DIC is very likely an important mechanism in coagulation defects found in extra-corporeal circulation. It may be significant that most problems in this regard are found in heart-lung by-pass (in contrast to just heart by-pass), where millions of small bubbles are an inevitable consequence of the membrane oxygenator. This situation is certainly reminiscent of decompression sickness.

CONCLUSIONS

Disseminated intravascular coagulation is an acute, transient coagulation occurring in the flowing blood throughout the vascular tree which may obstruct the microcirculation. As described by Hardaway,[58] DIC occurs in a variety of disease states, however, shock is its most typical feature.

A research of the literature has shown for the first time that DIC probably occurs in decompression sickness, especially in cases of "decompression shock" or "decompression collapse syndrome." It may occur alone or in conjunction with intravascular bubble formation. In most cases DIC probably precipitates intravascular bubble formation, but in some cases the bubbles themselves may be responsible for perpetuating DIC.

8

More studies are needed to fully elucidate the role of DIC in decompression sickness, however, this is an important new approach and it does help to explain many of the inadequacies of the bubble theory. The occurrence then of DIC in decompression sickness does not dispute the bubble theory. It is an addition (rather than a substitution) which makes it more explainable.

At preesnt, the most successful adjunct to recompression therapy is Dextran, and its known mechanisms of action are intimately concerned with reversing the conditions which precipitate DIC. Hopefully, other new adjunctive forms of therapy will follow. It seems likely that recompression will continue to be the mainstay in treating decompression sickness, however, sole reliance should not be placed on any single form of therapy.

BIBLIOGRAPHY AND REFERENCES

1. Adler, H. F.: Neurocirculatory collapse at altitude. U.S. Air Force School of Aviation Medicine; Randolph Field, Texas, Special Project, June. 1950.

2. Aggazzotti: Azione dell'aria compressi sugli animali. Il tempo di coagulazione del sangue. Boll. Soc. Ital. Biol. Sper., 8:180, 1933.

3. Anderson, H. T.: Factors determining the circulatory adjustments to diving. Acta Phys. Scand., 58:173, 1963.

4. Anderson, H. T.: The reflex nature of the physiological adjustments to diving and their afferent pathway. Acta Phys. Scand., 58:263, 1963.

5. Anon: A historical review of the bubble theory of the etiology of decompression sickness as related to high altitude exposure. Aeromed. Rev., 10-65 (AD 628751).

6. Archambeault, C. P.: A fatal case of caisson disease. Nav. Med. Bull., Wash. 19:167-168, 1923.

7. Baker, R. J., Shoemaker, W. C., Suzuki, F., et al.: Low molecular weight Dextran therapy in surgical shock. Arch. Surg., 89:373-389, 1964.

8. Barnard, E. E. P., Hanson, J. M., et al.: Post-decompression shock due to extravasation of plasma. Brit. Med. J., 2:154-155, 1966.

9. Barnard, E. E. P.: Decompression sickness; paradoxical response to recompression therapy. Brit. Med. J., 2:809-810, 1966.

10. Barthelemy, L.: Blood coagulation and chemistry during experimental dives and the treatment of diving accidents with heparin. pp. 46-56 in: Second Symposium on Underwater Physiology. Edited by C. J. Lambertsen and L. J. Greenbaum, Jr., N. R. C., Wash., D.C., N. R. C. Publication 1181, 1963.

11. Behnke, A. R.: Decompression sickness. Mil. Med., 117:257-271, 1955.

12. Behnke, A. R.: Symposium on industrial medicine: effects of high pressure: prevention and treatment of compressed air illness. Med. Clin. N. Am., 26:1213-1237, July, 1942.

13. Berg, W. E., et al.: Additional mechanisms for the origin of bubbles in animals decompressed to similated altitudes. J. Gen. Phys., 28:235, 1945.

14. Bergentz, S. E., Eiken, O., and Nilsson, I. M.: Effect of Dextran of various molecular weight on the coagulation in dogs. Thromb. Diath. Haemorrh., 6:15-24, 1961.

15. Bert, P.: Barometric pressure. Translated from French by M. A. and F. A. Hitchcock. College Book Co., Columbus, Ohio, 1943.

16. Bert, P.: Sur l'influence des modifications dans la pression atmospherique. C. R. Soc. Biol. Paris, Ser. 5, 5:27-30, 1873.

17. Bert, P.: Sur les effets des modifications de la pression barometrique. Gaz. Med. Paris, Ser. 428:386-387, 1873.

18. Bigelow, W. G., Heimbecker, R. O., and Harrison, R. C.: Intravascular agglutination of erythrocytes (sludged blood), vascular stasis, and the sedimentation rate of blood in trauma. Arch. Surg. 59:667-690, 1949.

19. Bloom, W. L., Harmer, D. S., Bryant, M. F., and Brewer, S. S.: Coating of vascular surface and cells: A new concept in prevention of intravascular thrombosis. Proc. Soc. Exp. Biol. Med., 115:384-386, 1964.

20. Bloom, W. L.: Present status of plasma volume expanders in the treatment of shock. Arch. Surg., 63:739-741, 1951.

21. Boycott, A. E., and Damant, G. C. C.: On the blood-volume of goats and its relation to their varying susceptibility of symptoms of caisson disease. J. Physiol., 36:xiv, 1907-08.

22. Boyle, R.: New pneumatical observations about respiration. Philos. Trans., 5:2011-2056, 1670.

23. Brestkin, M. P. (ed.): The effect of the gas medium and pressure on body functions, III (translated from Russian).

24. Brick, I.: Circulatory response to immersing the face in water. J. Appl. Physiol., 21:33, 1966.

25. Brunner, F. P., Frick, P. G.: Post-decompression shock due to extravasation of plasma. Lancet, 1071-1073, 16 May, 1964.

26. Carey, J. S., Brown, R. S., Woodward, N. W., et al.: Comparison of hemodynamic responses to whole blood and plasma expanders in clinical traumatic shock. Surg. Gynec. & Obst., 121: 1059-1065, 1965.

27. Catchpole, H. R., and Gersh, I.: Pathogenic

factors and pathological consequences of decompression sickness, Physiol. Rev., 27:360-397, 1947.

28. Chrysanthou, C., et al.: Studies on dysbarism, II: Influence of bradykinin and 'bradykinin-antagonists' on decompression sickness in mice. Aero. Med., 741-746, Aug, 1964.

29. Clarke, Liberman, Nyboer, Tapperman: Peripheral circulation during decompression: Digital volume pulse. CAM Rep. #232, 29 Nov, 1948.

30. Clay, J. R.: Histopathology of experimental decompression sickness. Aero. Med., 34(12):1107-1112, 1963.

31. Coburn, K. R., et al.: Decompression collapse syndrome. Aero. Med., 33:1211-1215, 1962.

32. Cockett, A. T. K., Nakamora, R. M., and Franks, J. J., et al.: Delayed shock in experimental dysbarism. Surg. Forum, 14:7-8, 1963.

33. Cockett, A. T. K., Nakamora, R. M., et al.: A new concept in the treatment of decompression sickness (dysbarism). Lancet, I:1102-1103, 1964.

34. Cockett, A. T. K., and Nakamora, R. M.: Newer concepts in the pathophysiology of experimental dysbarism—decompression sickness. Amer. Surg., 30(&):447-451, (July) 1964.

35. Cockett, A. T. K., Nakamora, R. M., and Franks, J. J., et al.: Recent findings in the pathogenesis of decompression sickness (dysbarism). Surgery, 58:384-389, 1965.

36. Cockett, A. T. K., Nakamora, R. M., and Kado, R. T.: Physiological factors in decompression sickness. Arch. Env. Health, II:760-764 (Dec), 1965.

37. Cockett, A. T. K., Kado, R. T., et al.: Altered pulmonary hemodynamics following experimental decompression sickness. Aerospace Med., 38(9):923-925, 1967.

38. Cook, S. F., Williams, O. L., Lyons, W. R., and Lawrence, J. H.: A comparison of altitude and exercise with respect to decompression sickness.

39. Cotes, J. E.: Decompression sickness with post-decompression collapse. Flying Personnel Research Committee Report #825, pp. 1-10, RAF Institute of Aviation Medicine, Farnborough, Hants, England, 1953.

40. Dewey, A. W., Jr.: Decompression sickness, an emerging recreational hazard. New Eng. J. Med., 267:759-765, 812-820, 1962.

41. Duffner, G. J.: Medical problems involved in underwater compression and decompression. Ciba Clinical Symposium, 10(4):100-120, 1958.

42. Elsner, R. W., and Scholander, P. F.: Selective ischemis in diving man. Am. Heart J., 65:571, 1963.

43. End, E.: Rapid decompression following inhalation of HeO₂ mixtures under pressure. Amer. J. Physiol., 120:712-718, 1937.

44. End, E.: The use of new equipment and helium gas in a world record dive. J. Industr. Hyg., 20:511-520, 1938.

45. Epstein, P. S., Plesset, M. S., et al.: On the stability of gas bubbles in liquid-gas solutions. J. Chem. Physica, 18(11):1505-1509, 1950.

46. Erdman, S.: The acute effects of caisson disease. Trans. from 15th Internat. Congr. Hyg. (Demogr.), Wash., 3(2):619-625, 1912.

47. Erdman, S.: The acute effects of caisson disease or aeropathy. Am. J. Med. Sci., 145:520-526, 1913.

48. Evarts, C. M.: Low molecular weight Dextran. Med. Clinics of N. Am., 51(5):1285-1299, 1967.

49. Evonuk, E., Sullivan, F. J., et al.: The effects of low molecular weight Dextran on cardiovascular and metabolic parameters in dogs. Arctic Aeromedical Lab., Ft. Wainwright, Alaska, AAO-TR-67-68, AD 655-070.

50. Gelin, L. E., and Zederfeldt, B.: Experimental evidence of the significance of disturbances in the flow properties of blood. Acta Chir. Scandinav., 122:336-342, 1961.

51. Gersh, I., Hawkinson, G. E., et al.: The formation and appearance of tissue and vascular gas bubbles after rapid decompression of guinea pigs from high pressure atmospheres. NMRI Research Project X-284, Report #2, 1944.

52. Ghose, N. N.: Death from compressed air sickness in India. Indian Med. Gaz., 65:698-699, 1930.

53. Glaser, E. M., et al.: Effects of heat and cold on the distribution of blood within the human body. Clin. Science, 9:181, 1950.

54. Golding, F. C., et al.: Decompression sickness during construction of the Dartford Tunnel. Brit. J. Indust. Med., 17:167-180, (Jul), 1960.

55. Goodman, M. W.: The syndrome of decompression sickness in historical perspective. Report #5 on Subtask (2) of BuMed Research Project MR005:14-3100, 21 Dec, 1961.

56. Gowdey, C. W., Philip, R. B., et al: Etiology and treatment of experimental decompression sickness with special reference to body lipids. Mil. Med. pp. 648-652, Jul, 1965.

57. Greenbaum, L. J.: A Bibliographical Sourcebook of Compressed Air Diving and Submarine Medicine, Vol. #3, Dec, 1966.

58. Hardaway, R. M.: Syndromes of Disseminated Intravascular Coagulation. Springfield, Ill., Charles C. Thomas, Publisher, 1966.

59. Hardaway, R. M.: New concepts of shock and its treatment. Clin. Med., 74:33-44, 1967.

60. Harding, F. E., et al.: Diving bradycardia in man. J. of Phys., 181:401, 1965.

61. Harvey, E. N.: Decompression sickness and bubble formation in blood and tissues. Bull. N.Y. Acad. Med., 21:505-536, 1945.

62. Halbouty, M. R., Heisler, J. J.: Severe neuro-circulatory collapse at simulated altitude. U.S. Armed Forces Med. J., 6:1363-1370, Sep, 1955.

63. Haymaker, W., and Davison, C.: Fatalities resulting from exposure to simulated altitudes in decompression chambers. J. Neuropath. Exp. Neurol., 9:29-56, 1950.

64. Haymaker, W., and Johnston, A. D., et al.: Pathology of decompression sickness: A comparison of the lesions in airmen with those in caisson workers and divers. Mil. Med., 117:285-306, 1955.

65. Heiberg: Autopsie d'un malade mort en sortant de l'air comprime. Gaz. Med. Paris, Ser, 5, 7: 540, 1878.

66. Heimbecker, R. O., and Bigelow, W. G.: Intravascular agglutination of erythrocytes (cludged blood) and traumatic shock. Surgery., 28:461-473, 1950.

67. Heimbecker, R. O., et al.: Role of gas embolism in decompression sickness—a new look at 'the bends'. Surgery., 64:1:264-272, 1968.

68. Heimbecker, R. O., et al.: The role of gas embolism in decompression sickness: A new look at the 'bends'. Abstract of presentation to the Society of Univ. Surgeons, scientific session, Statler Hilton Hotel, N. Y., N. Y., 10 Feb, 1968.

69. Hill, L. E.: The influence of atmospheric pressure on man. Lancet, 2:1-4, 1905.

70. Hill, L. E., and Greenwood, M.: The influence of increased barometric pressure on man No. 4. The relation to age and body weight to decompression effects. Proc. Roy. Soc., 80:12-24, 1907-08.

71. Hill, L. E., and Greenwood, M.: On the formation of bubbles in the vessels of animals submitted to a partial vacuum. J. Physiol., 39:xiii, 1909-10.

72. Hill, L. E.: The physiology and pathology of work in compressed air. Lancet, 1:575; 792, 1909.

73. Hill, L. E.: Compressed air illness and experimental research. Brit. Med. J., 1:348-353, 1912.

74. Hoff, E. C.: A Bibliographical Sourcebook of Compressed Air, Diving and Submarine Medicine, Vol. #1, Feb, 1948.

75. Hoff, E. C.: A Bibliographical Sourcebook of Compressed Air. Diving and Submarine Medicine, Vol. #2, Nov 1954.

76. Holmstrom, F. M. G.: Collapse during rapid decompression. J. Avia Med., 29:91-96, 1958.

77. Hornberger, W.: Decompression sickness. German Aviation Med., World War II, Vol. I. Prepared under auspices of Surgeon General of the U. S. Air Force, Wash., D. C., pp. 354-394, 1950.

78. Irving, L.: Changes in the blood flow through the brain and muscles during the arrest of breathing. Amer. J. Phys., 122:207, 1938.

79. Jacobs, M. H., and Stewart, D. R.: Observations on the blood of albino rats following rapid decompression. U. S. NRC-CAM Rept. #76, Oct, 1942.

80. James, P. M., and Hardaway, R. M.: The evaluation and therapeutic management of refractory shock in man. Mil. Med. 132(6): 419-425 (June) 1967.

81. Jullien, Leandri, and Crozat: Essai de verification biologique des lois physiques de decompression apres un sejour dans l'air comprime. From the laboratoire d'hygiene et de Medecine Sociale de la Faculte de Medecine de Marseille.

82. Kabat, H., and Levine, M.: Capillary emboli as lethal factor in burns. Science, 96:476, 1942.

83. Kemph, J. P., and Hitchcock, F. A.: Changes in the blood following explosive decompression. Amer. J. Phys., 155:477, 1948.

84. Kern, J. D.: The etiology and pathological physiology of decompression sickness. SubMed ResLab Report No. 345, Dec., 1960.

85. Kindwall, E. P.: Metabolic rate and animal size correlated with decompression sickness. Amer. J. Physiol., 203:385-288, 1962.

86. Knisely, M. H., et al.: Sludged Blood. Science, 106:431-440, 1947.

87. Kratochvil, C. H.: Contribution of Alphonse Jamminet to understanding of decompression sickness. J. Aviation Med., 31:239-248, 1962.

88. Laborit, H. L., Barthelemy, L., and Perrimond-Trouchet, R.: Action de l'heparine dans le traitement des accidents de decompression. Rev. Agressol., 2:229-235, 1961.

89. Lamphier, T. A., et al.: Decompression sickness. Indust. Med., 31:239-248, 1962.

90. Leverett, Bitter, McIver, et al.: Studies in decompression sickness: circulation and respiratory changes associated with decompression sickness in anesthetized dogs. SAM-TDR 63-7. AD402148, Mar., 1963.

91. Lyle, C. B.: Protection of rapidly decompressed rats by pharmacologic and physical means. Amer. J. Physiol., 201(5):759-761, 1961.

92. Malette, W. G., Fitzgerald, J. B., Cockett, A.T.K., et al.: Dysbarism—review of 35 cases with suggestion for therapy. Aerospace Med. 30(11):840-846, 1959.

93. Masland, R. L.: Review of cases of collapse occurring in altitude chambers. AAF, School of Aviation Medicine, Randolph Field, Tex., Special Project, June, 1950.

94. McCally, M.: Body fluid volumes and the renal response of human subjects to water immersions. Aerospace Med. Div., Wright-Patterson AFB, Ohio; processed for DDC-AD623724.

95. Nair, C. S.: Effects of breathing 100% O_2 at ground level on blood clotting time and platelet count. Ind. Jour. Med. Res., 55:123-127 (Feb) 1967.

96. Odland, L. T.: Fatal decompression illness at an altitude of 22,000 feet. Aerospace Med. 30 (11):840-846, 1959.

97. Oliver: Man suffering from caisson disease or compressed air illness. Northumb. Durh. Med. J., 7; 8-11, 1899.

98. Paulev, P.: Decompression sickness following repeated breath-holding dives. J. Appl. Phys., 20:1028, 1965.

99. Peterson, L. H.: Cardiovascular performance underwater. Proceedings Second Symposium on Underwater Physiology, p. 267, 1963.

100. Pfrommer, J. R.: Decompression sickness: The state of the art. U.S. Armed Forces Med. J., 10:1292, 1959.

101. Philips, R. E.: The ameliorative effect of heparin and depolymerized hyaluronate on decompression in rats. Can. J. Physiol. & Pharm., 42:319, 1964.

102. Poiseuille: Effect of high atmospheric pressures on capillary circulation. C. R. Acad. Sci., Paris, 13:933-934, 1841.

103. Rait, W. L.: Post-decompression shock. Med. J. Aust. 11 Oct., 1952.

104. Rivera, J. C.: Decompression sickness among divers: An analysis of 935 cases. Mil. Med., 129:314-334, 1964.

105. Robie, R. R., Lovell, F. W., and Townsend, F. M., et al.: Pathological findings in cases of decompression sickness. Aerospace Med., 31:885, 1960.

106. Rodbard, S.: Recurrence of decompression sickness on descent from high altitudes. J. Aviation Med. 17:89-91, 1946.

107. Schaefer, K.: Circulatory adaptations to the requirements of life under more than one atmosphere of pressure. Circulation III. Chap. 51, p. 1843, 1965.

108. Schaffer, E.: Sektionsbefunde bei Pressluft—(caisson) Arbeitern, 2. MedBeamt., 11:389-397, 1898.

109. Schubert, G., and Gruner, A.: Die Entstehung freier Gase in Blut und Geweben bei rascher Dekompression. Klin. Wschr., 18:988-990, 1939.

110. Schumer, W.: The microcirculatory and metabolic effects of dibenzyline in oligemic shock. Surg. Gyn. Obst., 123(4):787-791.

111. Shaw, L. A.: The physiological effects of high pressures. J. Industr. Hyg., 18:486-496, 1936.

112. Shilling, C. W.: Compressed air illness. Nav. Med. Bull., Wash., 39:367-376, 1941.

113. Shimoyama, M.: Cases of caisson disease with a fatal case caused by derangement of diving-bell. Bull. Nav. Med. Assoc., Japan, 19(5):44-48, 1930.

114. Shires, T., and Carrico, C. J.: Current state of the shock problem. Curr. Probl. Surg. pp. 3-67, Mar., 1966.

115. Swindle, P. F.: The possible relationship between intravascular agglutination of red blood cells and decompression sickness. U.S. NRC CAM Report #179, Aug. 13, 1943.

116. Swindle, R. F.: Occlusion of blood vessels by agglutinated red blood cells, mainly as seen in tadpoles and very young kangaroos. Amer. J. Physiol., 120:59-74, 1937.

117. Thomson, T. K.: An unsuspected cause of caisson disease. Int. Cong. Hyg. (Demogr.) 15(3): 608-610. 1912.

118. Thost, A.: Uber Caisson Krankheit. Fortschr. Med., 27:69-71, 1909.

119. Van Rensselaer, H.: The pathology of the caisson disease. Trans. Med. Soc. St. N.Y., pp. 408-444, 1891.

120. Wagner, C. E.: Observations of gas bubbles in pial vessels of cats following rapid decompression from high pressure atmospheres. J. Neurophysiol. 8:29-32, 1945.

121. Webster, D. P.: Skin and SCUBA diving fatalities in the U. S. Pub. Health Rep., 81:703-711, 1966.

122. Welham, W., Waite, C. L., et al.: Decompression sickness: Report of two unusual cases. U.S. Armed Forces Med. J., 2(8):1201-1204 (Aug) 1951.

123. Whitaker, D. M., Blinks, S. R., Berg, W. E., Twitty, V. C., and Harris, M.: Muscular activity and bubble formation in animals decompressed to simulated altitudes. J. Gen. Physiol., 28:213-223, 1945.

124. Whitehorn, W. V., Lein, A., and Hitchcock, F. A.: Changes in the blood following explosive decompression. Amer. J. Physiol, 155:477, 1948.

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author)	2a. REPORT SECURITY CLASSIFICATION
U.S. Naval Submarine Medical Center, Submarine Medical Research Laboratory	UNCLASSIFIED
	2b. GROUP

3. REPORT TITLE

DISCUSSION OF DISSEMINATED INTRAVASCULAR COAGULATION IN DECOMPRESSION SICKNESS

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*

Submarine medical officer thesis

5. AUTHOR(S) *(First name, middle initial, last name)*

John A. HOLLAND, LCDR, MC, USN

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
20 June 1969	12	124

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
	SubMedResLab Report No. 585
b. PROJECT NO. MF099.01.01	
c.	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*
d.	

10. DISTRIBUTION STATEMENT

This document has been approved for public release and sale; its distribution is unlimited.

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	U.S. Naval Submarine Medical Center Box 600, Naval Submarine Base Groton, Conn. 06340

13. ABSTRACT

The etiology of decompression sickness is disucussed from the point of view of its historical development from 1670 (Robert Boyle) to the present. A number of authors are cited who have called attention to circumstances unexplained by the bubble theory, and pointing to the existence of other mechanisms in the production of decompression sickness.

The concept of disseminated intravascular coagulation (DIC) is one of the mechanisms which has been receiving much attention as a possible coexisting condition in at least some of the cases of decompression sickness. DIC is a transient coagulation occurring in the flowing blood throughout the vascular tree which may obstruct the microcirculation. It involves the transformation of fibrinogen to fibrin; it includes the agglutination of red blood cells, and sticking of platelets. It is often seen in cases of shock.

Further development of these theories may lead to new forms of therapy for decompression sickness.

An extensive bibliography (124 items) is included.

14 KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Decompression sickness Bubble theory Etiology of decompression sickness Theory of blood coagulation Disseminated intravascular coagulation						